A case of vivax malaria with splenic infarction

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Splenic infarction occurs most commonly in the setting of hematologic disorders like sickle-cell anemia, leukemia, lymphoma, polycythemia vera and hypercoagulable states like protein C, protein S deficiencies due to clogging of the splenic microcirculation by abnormal cells¹. Other conditions include thromboembolic phenomenon like infective endocarditis, atrial fibrillation and postcardiac surgery. In malaria, splenic infarction is a rare complication and most of the reported cases of splenic infarct in malaria have been due to infection with *Plasmodium falciparum* and is less common with *P. vivax*.^{2–5} We report this case for its rarity.

Case Report

A 40 yr-old male presented with high grade, intermittent fever with chills and rigor for 2 wk and abdominal pain localised to left hypochondriac region for 4 days. The pain was continuous, not severe and not radiating to chest, left shoulder or loin. There was no history of jaundice, hematemesis, melena, cough or dyspnoea. The past medical history was insignificant. His vital parameters were BP-90/60 mmHg, pulse was 100/min regular. There was no pallor, but mild icterus was present. On per abdomen examination, tenderness and guarding was noted in the left hypochondriac region extending to the left lumbar region. The liver was palpable 2 cm below right costal margin and spleen was also palpable 3 cm below the left costal margin, soft and tender.

Peripheral smear showed schizonts of *P. vivax* with platelet count of 76000/mm³. There were no ring forms or gametocytes. *Plasmodium* LDH (lactate dehydrogenase) immunochromatographic test was positive for *P. vivax*. PfHRP2 (*P. falciparum* histidine rich protein 2 antigen) test was negative. Other routine investigations were with normal limits.

Ultrasound of the abdomen showed mild fatty changes of the liver, spleen of 16.5 cm with mild heterogeneity in upper pole with hypoechoic lesion. Patient was treated with intravenous artesunate 120 mg at 0, 12 and 24 h, followed by 120 mg daily for 7 days. Oral doxycycline 100 mg twice daily was given for 7 days. For the abdominal pain intravenous pantoprazole 40 mg once daily was given, along with intravenous tramadol on prn basis and nasogastric tube aspiration. On Day 3 of admission a contrast enhanced computed tomography (CT) of the abdomen was taken as the patient continued to have abdominal pain. Figs. 1 a & b show enlarged spleen with a large non-enhancing hypoattenuating area involving the





Fig. 1 (a) & (b): Contrast enhanced CT scan of the abdomen during arterial phase (a); and late venous phase (b) show enlarged spleen with a large non-enhancing hypoattenuating area (arrow) involving the anterior aspect of spleen consistent with splenic infarct.

anterior aspect of spleen consistent with splenic infarct. The patient was monitored for further complications like splenic rupture by closely recording the vital signs, six hourly abdominal girth charting, and for any worsening of abdominal pain. Patient responded well to artesunate and doxycycline, and became afebrile and abdominal pain subsided, nasogastric tube was removed on Day 6 when oral fluids were started. Platelet count returned to normal. The splenic infarction was managed conservatively as outlined above, without need of surgical intervention. A CT of the abdomen was taken 5 months later showed only irregular contour of the spleen on the anterior aspect.

Classically *P. vivax* causes an acute febrile illness and usually has a benign course with no complications or death compared to *P. falciparum*. Severe malaria is usually caused by *P. falciparum*. But complications occurring with *P. vivax* are being increasingly reported and it has been suggested that *P. vivax* infection should no longer be considered benign^{6, 7}. The complications that are seen with *P. vivax* are similar to those seen with *P. falciparum*. These include thrombocytopenia, anemia, liver dysfunction, respiratory involvement, renal impairment, circulatory collapse, and cerebral involvement⁸. Splenic infarct, splenic rupture and organ failure are uncommon complications.

The patient in this case had thrombocytopenia and symptomatic splenic infarct. Thrombocytopenia is one of the common complications seen in *P. vivax* malaria, whereas symptomatic splenic infarct is a rare complication of P. vivax^{1,2}. A search on PubMed for 'malaria' and 'splenic infarction' showed 8 cases of splenic infarct in P. $vivax^{9-14}$. In these reported cases, most patients had left upper quadrant abdominal pain, left pleuritic chest pain and tender splenomegaly. The diagnosis of splenic infarct was confirmed either by ultrasound or by CT abdomen. Probably splenic infarct in P. vivax malaria is an under reported complication. In a retrospective review from South Korea of 34 cases of *P. vivax* malaria which had undergone CT abdomen for gastrointestinal indications, it was found that 13 patients (38%) had splenic infarct¹⁵.

The occurrence of splenic infarct in *P. falciparum* is explained by the ability of its mature trophozoite and schizont forms to sequester in the deep venous microvasculature, whereas sequestration is not a feature of *P. vivax*. What could have possibly led to splenic infarction in this patient is sudden enlargement of the spleen in response to overwhelming infection with subsequent hypoxic injury.

Spontaneous splenic rupture is a known rare complication of *P. vivax*, which occurs during acute infection and during the primary attack¹⁶. Chronically enlarged spleens are less vulnerable to rupture. Other splenic complications reported in malaria are splenic hemorrhage, splenic abscess and splenic torsion^{17–18}. Therefore, clinicians should consider splenic complications like splenic infarct, spontaneous splenic rupture and hemorrhage in the differential diagnosis of left upper quadrant pain in a patient with malaria. It is possible that many cases of splenic infarcts in malaria are missed because ultrasound and CT of the abdomen are not routinely done in patients of malaria.

The treatment of splenic infarct is conservative with good outcome, as documented in our patient also. The possible complications of splenic infarct are of splenic abscess, rupture and hemorrhage¹⁹, to which the clinician should be vigilant, and which may require surgical intervention.

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